Risk of Connective Tissue Injury in Dysmenorrheic vs. Eumenorrheic Collegiate Athletes: A Case-Control Study

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INTRODUCTION

In recent years females have become increasingly engaged in athletic activities [1]. It is generally accepted that these young women are more likely to benefit from participation than be harmed, since exercise is often associated with many health benefits [1, 2] as well as greater academic success and the avoidance of high-risk behaviors such as drug, alcohol, and tobacco abuse [3]. However, for some females, exercise is also associated with a unique set of problems. For example, the sensitive female reproductive system can be largely impacted by the hormonal changes associated with exercise and energy balance. Females also gain over half of their adult skeletal mass and 15% of their adult height during adolescence, so exercise-induced changes in bone mass accretion can impact future skeletal health [3, 4]. The benefits of exercise may be trumped by negative consequences when a female exercises without properly fueling herself, and the likely results are changes in menstrual cycle and possibly bone mass.

Exercise-induced menstrual cycle changes, decreased bone mass and disordered eating issues are often discussed as the Female Athlete Triad [2, 5]. This term was coined in 1992 at a conference of The Task Force on Women's Issues of the American College of Sports Medicine [2, 5, 6]. It is most notable in sports where low body weight and/or a lean figure are thought to be beneficial to performance, since athletes are more likely to feel pressure to achieve an "ideal body weight" [6]. The existence of the female athlete triad has recently come under question [2, 3]. Some of the issues with identifying existence of the triad likely relate to the difficulty in measuring diet as well as the cost involved in measuring menstrual cycle function and bone density.

Although these complexities make identification of the triad challenging, it is important to examine relationships between the components to help identify how to focus diagnostic, education and prevention efforts. The current study evaluates the risk of self-reported ligamentous and boney injuries in female athletes with self-reported menstrual dysfunction compared with their cohorts with apparently regular menstrual cycles.

LITERATURE REVIEW

Most research suggests a higher risk of the female athlete triad in "lean sports". Examples of these sports include activities with subjective scoring (dance, figure skating, diving, gymnastics, aerobics), endurance sports (distance running, cycling, cross country skiing), sports requiring revealing clothing during competition (volleyball, swimming, diving, cross country running/skiing, track, cheerleading), sports utilizing weight classes (horse racing, wrestling, rowing), and finally, sports emphasizing pre-pubertal physique (figure skating, gymnastics, diving) [2]. Athletes in lean sports may be more susceptible to the triad due to continually suppressing body weight in an effort to maintain a lean appearance.

It is difficult to ascertain the true prevalence of the triad because eating disorders and menstrual function are private issues, so the triad is likely underreported thus overlooked. For example, the loss of menses is often viewed by athletes or coaches as an indicator of peak training level rather than a sign of overtraining or energy imbalance, so it is often discounted or even applauded [7]. Many of the negative consequences of amenorrhea and the triad may be prevented if coaches, trainers and parents become more informed

and aware of the symptoms and potential consequences of amenorrhea. Efforts to increase awareness that amenorrhea is a potential indicator of harm and is not desirable should be at the forefront of the athletic healthcare staff's agenda.

The disordered eating component consists of clinical eating disorders as well as a whole range of harmful subclinical behaviors. At one end of the spectrum are anorexia nervosa and bulimia nervosa, which have specific criteria for formal diagnoses [8]. Somewhat less specific are any type of food restriction, fasting, binging, and purging using laxatives, vomiting, diuretics, or diet pills. Although often undiagnosed or unnoticed, preoccupations with food, exercise, or body image round out this spectrum of disordered eating. The nebulous signs of eating disorders may affect up to 62% of female athletes depending on the criteria for inclusion [3]. It is also important to acknowledge that a negative energy balance can be considered disordered eating whether or not it is intentional. Potential consequences not only impair performance, but increase the risk of injury and death, and include depleted lean body mass and glycogen stores, chronic fatigue, micronutrient deficiencies, electrolyte and acid-base imbalance, and cardiac and gastrointestinal problems [3]. Properly fueling the body consistent with the activity level demonstrates a healthy attitude towards eating and body image.

In order to identify menstrual dysfunction, a normal menstrual cycle must first be defined. A normal cycle begins with the follicular phase which is marked by the first day of menstruation. It lasts approximately 14 days, during which follicle-stimulating hormone (FSH) promotes the growth of a single follicle (egg), which secretes estradiol

and positively feeds back to simulate hypothalamic secretion of luteinizing hormone (LH.) The follicular phase ends with ovulation, when a large spike in LH level occurs. The next fourteen days make up the luteal phase, in which increased production of estradiol and progesterone negatively feed back to the hypothalamus to suppress LH and FSH release [7]. In normal menstrual function, or eumenorrhea, these cycles occur 10-13 times each year [9-12].

The role of the hypothalamus in normal menstrual function is critical. Since many hypothalamic releasing factors and hormones are involved with the menstrual cycle, the cycle can be perturbed by the impact of mental, physiological, or metabolic stressors on the hypothalamus. Because these stressors influence the menstrual cycle, it is important to consider early onset of training, intense training, and high mental stress when evaluating menstrual dysfunction [4, 7]. The lack of estrogen production is the likely outcome of these stressors and results in a hypoestrogenic environment that may impact bone and other tissues of the body.

Female menstrual dysfunction affects 6-79% of athletes, is multi-factorial, usually hypothalamic in origin, and may be associated with decreased bone mineral density (BMD) [1, 2]. As with disordered eating, menstrual dysfunction encompasses a large range of disorders. The most serious form of menstrual dysfunction is primary amenorrhea, which is defined as delayed menarche (no menses by age 16 in females with secondary sex characteristics.) [2, 6, 13] Secondary amenorrhea, the more common form of dysfunction associated with athletes, is defined as the absence of three or more

consecutive menses after menarche [2, 6, 13]. Another category along the spectrum of menstrual disturbance is oligomenorrhea, which is defined as three to less than six or eight cycles per year, depending on the study [14]. Amenorrhea is most generally recognized as the lack of a menstrual cycle or fewer than three cycles per year. Dysmenorrhea is often used when the cycle is irregular and in this study will identify the group of women having fewer than six cycles per year. In this study, dysmenorrhea includes oligomenorrhea and amenorrhea. Other menstrual disturbances are not diagnosed using the frequency of menstruation, but are still based on disrupted function. The least detectable and therefore the most underreported types of dysfunction are anovulation (no ovulation but bleeding may still be present) and shortened luteal phases (ovulation may occur but insufficient level of progesterone for endometrial development) [14]. Menstrual function category in this study is simply gauged by the self-reported frequency of menstruation as no hormonal tests were performed.

The final component of the Triad, osteoporosis (as well as osteopenia), is defined as low bone mass and deterioration of bone tissue leading to skeletal vulnerability and increased risk of injury [2]. This breakdown occurs as a result of premature bone resorption by osteoclasts and/or decreased bone formation by osteoblasts [3]. In the normal process of bone remodeling, the small fibers of trabecular bone break or microfracture then reform in response to growth and stress. Estrogen reduces bone resorption directly by inhibiting osteoclasts. In the hypoestrogenic condition, suggested by lack of a normal menstrual cycle, the resorption phase of normal remodeling is likely faster due to this osteoclastic disinhibition. The microfractures that normally occur with training may not repair at the

same pace they accumulate, and an insufficiency fracture may be more likely to occur [15].

Bone mass is usually measured by dual x-ray absorptiometry (DXA) and expressed in bone mineral density or BMD (grams/cm^2) to account for the size of the individual. It is common to categorize BMD in standard deviations relative to the average values for young adults of the same gender. The World Health Organization criteria for defining bone status are: normal (BMD within 1 SD below the mean for young adults), osteopenia (BMD 1-2.5 SD below the mean for young adults), osteoporosis (BMD >2.5 SD below the mean for young adults), and severe osteoporosis (BMD >2.5 SD below the mean for young adults and diagnosis of one or more fragility fractures) [2, 16]. Originally, the female athlete triad included formal osteoporosis, but recognition of the possible role of osteopenia in a less than optimal spectrum of skeletal mass has resulted in much debate about the degree of mineral disturbance that should be included in the triad. Beals *et al* recently categorized BMD <-1.0 SD as "at-risk for low BMD," and included it in the analysis even though it doesn't fit the formal criteria for osteoporosis [6]. Weight-bearing exercise is presumed to strengthen bone, but under hypoestrogenic conditions, the bone mass is often less than expected. Some current debate centers around including less than optimal bone mass as a criteria for the female athlete triad.

Though the three components of the triad have been identified, the likely mechanism is less clear. Although cross-sectional studies have shown an association between the disorders of the triad, a lack of longitudinal studies and well-controlled clinical

interventions makes it difficult to determine an etiological pattern [6]. For example, an early hypothesis by McArthur and Frisch suggested that 17% body fat was needed to induce menarche and 22% body fat was needed to maintain reproductive health [17-19]. It has since been confirmed that even though low body fat may be associated with disrupted reproductive dysfunction, it does not appear to be a direct mechanism since regular cycles have been observed in athletes with less than 17% body fat and athletes with identical body fat percentages can be either eumenorrheic or amenorrheic [20]. Matkovic has proposed a threshold of serum leptin (12.2 ng/mL) as a second messenger from the fat cells to the hypothalamus to initiate menarche, but this same threshold has not been proven for continued menses [21]. In a study of healthy, regularly menstruating females, Loucks and Thuma proposed a threshold of energy availability (30 kcal/kg lean body mass) necessary to sustain LH function and suggest the availability of glucose to the brain is the critical factor [22]. They have identified LH pulsatility disruption at a level of carbohydrate availability between 90 and 130 g/d of glucose [22]. Lastly, Loucks and Thuma noted that the effects of the spectrum of restricted energy availability on LH pulse frequency and amplitude most closely resembled incremental curves for plasma glucose, GH and cortisol patterns. The etiology of athletic amenorrhea remains somewhat elusive and is likely a combination of factors generated from the hypothalamus and the pituitary axes for the ovaries, adrenals and thyroid.

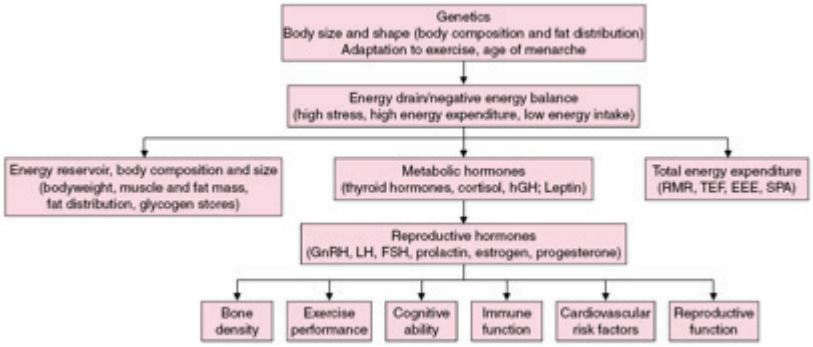

Figure 1. A model illustrating the influence of energy drain and high stress on the development of menstrual dysfunction in active women, and the potential health and performance outcomes due to low reproductive hormones and high cortisol levels: **FSH** = follicle-stimulating hormone; **GnRH** = gonadotropin-releasing hormone' **hGH** = human growth hormone; **LH** = luteinizing hormone; **RMR** = resting metabolic rate; **SPA** = spontaneous physical activity; **TEF** = therapeutic effect of food. Model taken from Manore *et al* [14]

Further application of this hypothalamic origin theory results in the most commonly accepted mechanism for the Triad and follows the "energy drain hypothesis" (Figure 1) [14]. The energy reservoir is the energy stored in glycogen and fat in addition to the energy consumed through food. Energy drain occurs when the energy reservoir is inadequate to support the daily energy expenditure [7]. According to Manore, most female athletes need 2,300 to 2,500 kcal/day to maintain body weight, while endurance athletes may need up to 4,000 kcal/day depending on mileage. A chronic negative energy balance is thought to lead to menstrual dysfunction by disrupting the hypothalamic-pituitary-ovarian (HPO) axis and the hypothalamic-pituitary-adrenal (HPA) axis [23].

Initially, the HPA axis, or "stress axis", is over-stimulated resulting in increased hypothalamic secretion of corticotrophin-releasing hormone, which increases pituitary release of adrenocorticotrophic hormone and finally, increases cortisol production by the adrenal cortex [7]. High levels of cortisol are counterproductive to skeletal and menstrual health and feed back to the HPO further suppressing biological function. The HPO axis,

or “reproductive axis”, is down-regulated by high levels of cortisol and the hypothalamic pulsatile release of gonadotropin-releasing hormone (GnRH) is suppressed. Low GnRH limits pituitary secretion of LH and FSH which in turn suppresses ovarian production of estradiol [13]. The absence of the LH/estradiol surge to mark the end of the follicular phase results in the suppression of menstrual cycles in affected athletes [1].

Once the level of endogenous estrogen production is chronically low, the athlete is likely to experience less than optimal bone modeling and formation [4, 9]. Depending on the skeletal site, peak bone mass is reached at age 18-25 and then lost at a rate of 0.3-0.5% annually after about age 30 [3]. However, hypoestrogenism during adolescence increases the risk to lose 2% of bone mass per year instead of gaining 2-4% while the bones are still consolidating. [3] This bone loss/suppression pattern is similar to that of postmenopausal women or women with a chronic hypoestrogenic condition such as premature ovarian failure, pituitary tumor, or anorexia nervosa [3]. In fact, a study of non-athletic Spanish women over 50 years old showed that a late age at menarche and/or presence of amenorrhea at any time during fertile period were associated with a higher incidence of osteoporotic fractures [24]. In athletes, important factors that may influence bone suppression include the length and severity of menstrual dysfunction, type of skeletal loading in the specific sport, nutrition, and genetics [3, 16].

Adult bone is a living tissue with a balance of formation and resorption at any given time. Decreased bone mass can be the result of decreased bone formation or increased bone resorption [16]. In a study of rhythmic gymnasts, bone age was delayed an average of

two years when compared with chronological age [25]. The study suggested this was likely caused by an increase in resorption rate with no change or balance in formation rate. Resorption was estimated by high levels of alpha-CTX (an isomer of the carboxy-terminal telopeptide of collagen I) as a biochemical indicator of bone resorption that is especially sensitive in adolescents. Slowed bone formation as well as stimulated bone resorption results in a less than optimal peak bone mass and this gymnast study demonstrates how a lack of that balance impacts final bone mass.

In a case-control study of 49 female athletes (29 amenorrheic and 20 eumenorrheic, as confirmed by serum estradiol and progesterone level tests), bone mass was estimated at several skeletal sites using DXA. Most of the women were competitive or recreational runners, and criteria for inclusion in the study included participation in some type of weight-bearing exercise, 45 minutes of activity 4 days a week, and no oral contraceptive use in the previous 6 months leading up to the study [10]. When comparing the BMD of amenorrheic and eumenorrheic athletes, the amenorrheic girls had significantly lower BMD at several sites, including the lumbar spine, femoral neck, trochanter, Wards triangle, intertrochanteric region, and shafts of the femurs and tibiae [10]. At every site, BMD correlated positively with body weight and negatively with duration of amenorrhea. Fifteen percent of eumenorrheic girls were osteopenic or osteoporotic compared to 72% of amenorrheic girls. As suggested in this study, weight-bearing exercise is not necessarily sufficient to offset the detrimental effects of low endogenous estrogen levels [10].

In another study, which matched 25 athletes with stress fractures to 25 control athletes with no history of bone injury, athletes with stress fractures had low BMDs in both the axial and appendicular skeleton, and at sites with varying ranges of trabecular to cortical bone ratios [12]. Subjects with stress fractures had a higher frequency of current menstrual irregularity and a lower occurrence of oral contraceptive use. It is also important to note that subjects were not recruited based on menstrual dysfunction, which strengthened the validity of the relationships between stress fractures, menstrual dysfunction, and oral contraceptive use.

Another possible mechanistic key player in the energy drain hypothesis is leptin, which is a hormone product of the obesity (ob) gene secreted by adipocytes. Leptin is a regulator of basal metabolic rate and fluctuates with fat stores and energy availability [1, 26]. Leptin positively correlates with BMI and the diurnal rhythm of leptin concentrations are suppressed if energy intake is low [1]. Interestingly, leptin receptors have been found on hypothalamic neurons that control the GnRH pulse generator, which indicates that leptin may signal the reproductive axis of low energy availability. Leptin receptors have also been found on bone so decreased leptin levels may also suppress bone mass directly [1].

Even though treatments such as hormone replacement therapy are believed to prevent further bone loss, the effects of previous bone loss may be irreversible [9]. Since the hypothetical mechanism for bone loss is an uncoupling between an abnormally high bone resorption rate and an unchanged bone formation rate, estrogen therapy may not be sufficient to regain bone mass as it only affects the osteoclastic role in modeling [16].

This is especially true if the loss has been sustained in trabecular bone (trunk, spine, pelvis, and upper femur) versus cortical bone (femoral neck, radius), since trabecular bone has a faster resorption rate [20]. It is well-accepted that trabecular bone seems to be more labile during hypoestrogenism, and this leads to increased fragility fractures at primarily trabecular sites [12, 20]. If bone suppression disturbs the connectivity of the trabecular bone, current research does not offer a treatment that seems to reconnect the trabeculae so the micro-architecture is lost permanently. Preventing long bouts of low estrogen is important to the infrastructure of trabecular bone.

Although most information is available regarding bone injury, it is equally important to consider the potential effects of menstrual dysfunction on connective tissues such as ligaments or tendons. For example, since anterior cruciate ligament (ACL) injury rates are 4-8 times higher in women than men, it's logical to examine the unique female hormonal cycle as a possible contributor [27]. According to Wojtys, estrogen has a direct effect on collagen metabolism and viability and neuromuscular performance can vary throughout the menstrual cycle insinuating estrogen may play a significant role in connective tissue injuries such as ACL [27].

In a study of 28 females who met the criteria for normal menstruation and non-contact ACL injury, researchers found that a disproportionate number of injuries occurred during the ovulatory phase of the cycle when estrogen levels are lower [27]. If hormonal rise and fall contributes significantly to ACL injuries in eumenorrheic females, the hormonal instability and suppression associated with the Female Athlete Triad could very likely be

a major contributor to ligament injury in women with menstrual dysfunction. Previous literature suggests that pre-menopausal women who had absent or irregular menses, while engaged in vigorous exercise programs, were at an increased risk for musculoskeletal injury[28].

Since the Female Athlete Triad has been associated with such serious skeletal and connective tissue implications, it is important to monitor athletes for menstrual dysfunction as a potential marker of injury risk. The purpose of this case-control study is to compare athletes with self-reported menstrual dysfunction to sport- and body size-matched eumenorrheic controls in order to compare the relative likelihood of skeletal and connective tissue injuries with the working hypothesis that females with menstrual disturbance have a higher risk of injury than their matched controls.

METHODS

The current multi-site study recruited athletes at The Ohio State University, University of North Texas, and University of Utah, and has been approved by the OSU Social and Behavioral IRB (protocol 2004B0231). Written consent and completed questionnaires were obtained at pre-season physicals as part of the pre-participation evaluation. Although there may have been slight variations in the manner by which athletes received the questionnaire, the assumption has been made that the methods used by University of North Texas and University of Utah had no significant effect on their results as compared to Ohio State.

At The Ohio State University, the consent form and questionnaire were presented to each athlete at their annual pre-season physical. In the absence of coaches, athletes who chose to participate filled out the survey on-site and returned it before leaving. After data from all three universities was compiled, a total of 662 varsity athletes (451 women, 207 men) had filled out the questionnaire, spanning 33 sports. Of 451 women, 45 had too much missing data to be considered for analysis. Athletes were automatically removed from the data pool if either of the primary variables used for matching, race or sport, were missing. This yielded 397 female athletes in the pool and included 21 sports.

The complete survey consisted of a Demographic Questionnaire, Menstrual History Questionnaire, Injury Assessment Questionnaire, Questionnaire for Eating Disorder Diagnosis (Q-EDD), Bulimia Test Revised, Mood Scale (PANAS-X), Beliefs About Attractiveness Scale Revised (BAA-R), Body Parts Satisfaction Scale (BPSS), Multidimensional Body-Self Relations Questionnaire (MBSRQ), and the Drive for Muscularity Scale (DMS). For the focus of this study, the Demographic Questionnaire, Menstrual History Questionnaire, and Injury Assessment Questionnaire were the primary data source (Appendix A). The results of the questionnaires were entered into an excel database and imported into SPSS version 15.0 for statistical analysis.

Each dysmenorrheic athlete (defined as 0-6 periods per year) was then paired with a eumenorrheic control athlete (defined as 10-13 periods per year) using the following algorithm of variables:

1. same school, sport and ethnicity

2. height (closest within 2 inches)
3. weight within 20 pounds.

Matching yielded 36 pairs of athletes. Because of the small sample size, McNemar's non-parametric test was used to evaluate the predictive power of connective tissue injury (defined as a contact or non-contact ACL injury, meniscal injury, IT band injury, sprain, or tendonitis) and boney injury (defined as broken tibia, foot, femur, or spine) on menstrual status. The overall apriori statistical significance was set at $p \leq 0.05$.

After the primary evaluation of paired data, additional exploratory statistics were performed using the full dataset of athletes previously identified as dysmenorrheic and eumenorrheic. This analysis excluded athletes with 7-9 cycles per year to clearly delineate eumenorrhea from dysmenorrhea. Three binary logistic regression models were evaluated stepwise to identify the most significant predictors of menstrual status, connective tissue injury, and boney injury as the dependent variables. Independent variables of interest included age, year in school, ethnicity, sport, height, weight, satisfaction with body, whether or not the athlete reported an eating disorder, age at menarche, and body mass index (BMI). T-tests or Chi-square distribution testing between groups were used to confirm identified differences.

RESULTS

The distribution of the paired data for eumenorrheic ($n = 36$) and dysmenorrheic ($n = 36$) athletes based on history of connective tissue and boney injury is presented in Table 1. After evaluation of these pairs using McNemar's non-parametric test, neither connective

tissue injury (p = 0.607) nor boney injury (p = 0.100) served as a significant predictor of menstrual status.

TABLE 1
Distribution of Subjects and McNemar's Test Results Based on Menstrual Status, Connective Tissue Injury, and Boney Injury

Menstrual Status	Connective Tissue Injury		Boney Injury	
	No	Yes	No	Yes
Eumenorrheic	21	15	23	13
Dysmenorrheic	19	17	24	12
McNemar's model p-value	0.607		0.100	

The continued exploration using stepwise binary logistic regression modeling demonstrated age at menarche as the only significant predictor in both the menstrual status model (p = 0.000) and bone injury model (p = 0.042). In the connective tissue injury model, sport (p = 0.001) was the only variable of interest that made a significant contribution to the model. These results are summarized in Table 2, with significant p-values in bold.

TABLE 2
Binary Logistic Regression Models to Identify Significant Predictors of Menstrual Status, Bone Injury, and Connective Tissue Injury

Variable of Interest	Menstrual Status Model		Bone Injury Model		Connective Tissue Injury Model	
	dependent= menstrual status Step 1	menstrual status = age at menarche + X Step 2	dependent = bone injury Step 1	bone injury = age at menarche + X Step 2	dependent = CT injury Step 1	CT injury = sport + X Step 2
Age	0.965	0.896	0.503	0.491	0.276	0.476
Year	0.370	0.541	0.452	0.486	0.153	0.156
Race	0.344	0.436	0.816	0.965	0.365	0.392
Sport	0.812	0.964	0.770	0.682	**0.001**	
Height	0.638	0.269	0.821	0.784	0.604	0.379
Weight	0.319	0.946	0.398	0.651	0.976	0.829
Satisfaction with Body	0.588	0.400	0.535	0.669	0.662	0.746
Eating Disorder*	0.963	0.946	0.614	0.702	0.351	0.572
Age at Menarche	**0.000**		**0.042**		0.412	0.289
BMI	0.105	0.327	0.179	0.402	0.472	0.476

*Whether athlete self-perceives an eating disorder

Additional statistics were used to confirm the role of age at menarche in the menstrual status and bone injury models by comparing eumenorrheic and dysmenorrheic females. An independent samples t-test confirmed that the average age of menarche for eumenorrheic athletes was 13.00 ± 1.42 years, while the average age of menarche for dysmenorrheic athletes was 13.90 ± 1.94 years ($p = 0.000$). Similar testing for the bone injury model demonstrated the average age of menarche for females without bone injury to be 13.04 ± 1.50 years, while the average age of menarche for athletes who had experienced bone injury was 13.50 ± 1.46 years ($p = 0.047$).

In finding sport to be the only significant predictor for the connective tissue injury model, we formed an ancillary hypothesis that the relationship would be strengthened if the sports were divided into 'contact' and non-contact' categories. Before investigating this theory, we first observed the distribution of all female athletes by team and whether they had suffered connective tissue injury. This distribution is presented in Table 3.

TABLE 3

Distribution of Athletes by Team and Connective Tissue Injury

Team	no CT injury	CT injury
Basketball	16	16
Cheerleading	13	7
Cross Country	2	4
Diving	2	3
Fencing	2	2
Field Hockey	12	6
Golf	13	4
Gymnastics	9	13
Ice Hockey	3	2
Lacrosse	0	3
Rifle	2	1
Varsity Rowing	16	5
Novice Rowing	22	12
Soccer	4	15
Softball	17	29
Swimming	22	18
Synchronized Swimming	6	7
Tennis	4	20
Track & Field	11	25
Volleyball	6	14

The classification system applied was based on the stratification used by the National Collegiate Athletic Association (NCAA) to evaluate sport-associated risk of injury. The scale ranges from 1-4, with sports classified as a four having the highest injury risk based on available multi-year sport injury surveillance data [29]. A four-level variable for injury risk was created, with the classifications presented in Table 4. Chi-square analysis of the stratified variable failed to demonstrate significant differences between sport risk categories and connective tissue injury ($p = 0.251$).

TABLE 4
Distribution of Sports Based on NCAA Criteria for Injury Risk

1	2	3	4
Golf Hockey Track Rifle	Crew Cross Country Fencing Softball Swimming Tennis Synch. Swimming Diving	Field Hockey Lacrosse	Basketball Soccer Volleyball Gymnastics

Lastly, we hypothesized that division of sports into 'lean' and 'non-lean' categories as characterized by Otis *et al* [2] would show an association between lean sports, which historically have a higher prevalence of the Female Athlete Triad, and self-reported menstrual dysfunction, connective tissue injury, and boney injury. The distribution of athletes and results from corresponding chi-square analyses are presented in Table 5.

TABLE 5
Distribution of Subjects Based on Sport Type, Menstrual Status, Connective Tissue Injury, and Boney Injury

Sport Type	Menstrual Status		Connective Tissue Injury		Boney Injury	
	Eumenorrheic	Dysmenorrheic	No	Yes	No	Yes
Non-lean Sport	163	17	77	103	168	12
Lean Sport	191	26	109	108	181	36
Chi-Square Test p-value	0.418		0.138		**0.003**	

DISCUSSION

By pairing dysmenorrheic athletes to matched controls, we likely decreased the power of the study and were unable to identify a significant relationship between menstrual status and boney or ligamentous injuries. We do believe that this relationship exists, but the scope and power of this study was likely not large enough to support our hypothesis. For example, the aforementioned Rencken study of 49 competitive and recreational runners showed that 15% of eumenorrheic girls were osteopenic or osteoporotic compared to 72% of amenorrheic girls [10]. With such strong evidence pointing towards the relationship between menstrual and skeletal health, larger scale studies must be conducted to better identify the existence of the triad.

In finding age at menarche to be a significant predictor for both the menstrual status and bone injury models using stepwise logistic regression with the full dataset, we concluded that an older age at menarche is associated with both future dysmenorrhea and bone injury. T-tests for age of menarche between the eumenorrheic and dysmenorrheic athletes as well as between females with and without bone injury confirmed this finding. Consequently, age of menarche should be considered when evaluating a teenage female athlete's risk for menstrual and/or musculoskeletal consequences.

After stepwise logistic regression with the full dataset identified sport as the only significant predictor for the connective tissue injury model, we investigated a resultant hypothesis that the association would be strengthened if the sports could be stratified according to risk criteria according to NCAA guidelines. Although this grouping did not

lead to a significant association between contact sports and increased risk of connective tissue injury, a more definitive classification system for the sports could result in an association in future studies.

After classification of the sports based on 'lean' vs. 'non-lean' criteria, the chi-square analyses showed a significant association between lean sports and bone injury. This finding reinforces previous studies which have shown that athletes in lean sports may be more susceptible to the female athlete triad due to an ongoing suppression of body weight in an attempt to maintain a trim appearance [2]. Therefore, classification of athletes based on sport type remains an important tool for predicting the likelihood of bone injury as well as menstrual status in future studies.

CONCLUSIONS

According to the results of this study, more research needs to be conducted regarding the influence of menarcheal age and risk of future injury and menstrual status. Similarly, sport type and intensity of training at an early age should also be considered in future research designs. Although we feel that paired case-control data is an excellent way to assess the association between menstrual dysfunction and bone or connective tissue injuries, future studies would likely require a larger number of paired subjects in order to identify significant relationships.

As it stands, the positive association between delayed menarche and musculoskeletal injuries indicates a greater need for education about the Female Athlete Triad among

athletes, coaches, trainers, parents, and healthcare professionals, including risks and warning signs of the Triad and the dangers of pushing athletes beyond safe training levels. [3] Increased knowledge and awareness of the Triad has the potential to benefit athletes by avoiding injury and offsetting the resultant long-term health consequences.

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APPENDIX A

Athlete Questionnaire

I. Background Information

1. Date of Birth _____

2. Subject ID _____

3. Current Academic Status: ___Freshman ___Sophomore ___Junior ___Senior ___5th Year

4. Race/Ethnicity: ___Caucasian/White ___Hispanic/Latino/Mexican-American ___American Indian
____African-American/Black ___Asian American/Pacific Islander ___Other_____

II. Sports History

1. Current sport _____
   a. Years of participation in current sport at current university:_____
   b. Overall years of participation in current sport: _____
2. Do you currently receive an athletic scholarship for participating in your sport? _____Yes _____No
3. Are you currently a "starter" (or first-string player) in your sport? _____Yes _____No
4. In the past three months, on average, how many hours per week have you spent in sport-related activity such as practice, conditioning, weight training?
5. Do you run or jog outside of your sport? Yes No
   a. If yes, approximately how many hours per week? _____hours
6. Do you do other endurance training outside of your sport (this includes steppers, elipticals, bikes)
   Yes No
   a. If yes, approximately how many hours per week? _____hours
7. Do you engage in other recreational sports besides your sport when in season?
   Yes No
   Please list the sports and approximate hours per week.

III. Weight History

1. Present height: _____ feet _____ inches
2. Present weight: _____ pounds
   a. Length of time at current weight: _____ (months)
3. Ideal weight: _____ pounds
4. My body frame is: _____Small _____Medium _____Large

5. Are you satisfied with your current weight? ____ Yes ____ No
   a. If NO, do you consider yourself to be: ____ overweight ____ underweight

7a. Lowest weight in past 2 years:_____ b. Highest weight in past 2 years:_____

8. Do you experience significant weight fluctuations (changes of more than 10% of your body weight)
   - during the season for your sport _____ Yes _____ No
   - during the off-season _____ Yes _____ No

If yes, please

explain:_____
____

9. Using the following scale, indicate how much pressure do you feel from each of the following groups/entities to achieve/maintain a certain body size/shape/composition and/or weight:

1	2	3	4	5	6	7
None at all						Extreme

a. Coaches	1	2	3	4	5	6	7
b. Family	1	2	3	4	5	6	7
c. Friends	1	2	3	4	5	6	7
d. Boyfriend/Girlfriend	1	2	3	4	5	6	7
e. Judges in Your Sport	1	2	3	4	5	6	7
f. TV Shows/Movies	1	2	3	4	5	6	7
g. Fashion/Style Magazines	1	2	3	4	5	6	7
h. Teammates	1	2	3	4	5	6	7

10. Have you ever been diagnosed or treated for:
a. Anorexia Nervosa?_____Yes _____ No b. Bulimia Nervosa? _____ Yes _____ No
11. Do you think you might have an eating disorder? _____ Yes _____ No _____Maybe

IV. Menstrual History

1. Have you ever had a menstrual period? _____Yes _____No
   a. If YES, how old were you when you had your first menstrual period? _____
   b. Did you participate in competitive sports before you started your periods? _____

2. How many menstrual cycles have you had in the *past 12 months*? _____

3. On average, how many menstrual cycles *per year* have you had since your first menstrual period? _____

4. On average, how many days are there between your menstrual cycles? _____

5. On average, how many days do your periods (bleeding) last? _____

6. Do your periods change when you are training for your sport (i.e., in season)? _____Yes _____No
   a. If YES, please explain :_____

7. Please describe any other menstrual irregularities or problems you have experienced: _____ _____

V. Injury Assessment Questionnaire

1. Have you had a significant ligament or bone injury? Yes No

2. Have you had any of your knee or ankle injuries surgically repaired? Yes No
   2a.What year did you have surgery and what joint?

3. Have you ever had a stress fracture or other broken bone?
   If yes, list the bone and year of each fracture you have had

Name of Bone	Stress fracture	Year	surgery
	Y N		Y N
	Y N		Y N
	Y N		Y N